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THE CAUSAL RELATION OF OBSTRUCTED CARDIAC CIRCULATION TO LYMPH STASIS.

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In my former contributions to the medical press,¹ on the congenital and acquired forms of occlusion and dilatation of lymph channels, I collated a number of cases, previously published by different reporters, to illustrate the causes and special characteristics of these forms of disease. In a few of the cases it was evident that the existent lymphangiectasis was attributable to interruption of the blood vascular currents either through the heart or lungs. I referred incidentally to those reports, but the illustrative cases have not, as far as is known to me, been collected and arranged in a manner to distinctly exhibit the relation of cause and effect subsisting between obstructed cardiac circulation and lymphangiectasia.

Before citing the cases relating to the direct proposition, it is necessary to refer to the predicate—that occlusion or stenosis of the thoracic duct, at or near its terminal extremity at the left subclavian vein, may, when the collateral circulation is not speedily and sufficiently established, produce dilatation or rupture of some part of the lymph vascular apparatus. This can be shown both by the results of experimentation and by post-mortem reports.

Morton² performed a number of experiments upon dogs by ligating the thoracic duct; in some the “receptaculum and in other the lacteals burst.” In every case there was extravasation of chyle. In Sir Astley Cooper’s³ experiments, when rupture occurred it took place at the receptaculum; and Dupuytren’s experiments upon horses were followed by nearly similar results. Cooper says it is not necessary to tie the duct. If the animal is fed with milk and the extremity of the duct is compressed

¹ New Orleans Med. and Surg. Jour.; and Congenital Occlusion and Dilatation of Lymph Channels. Wm. Wood & Co., 1878, New York.

² Donald Monro, Essay on Dropsy and its Different Species, pp. 22, 23.

³ Medical Records and Researches, p. 100 *et seq.*

one-half hour afterwards, the receptaculum will rupture. Those and similar experiments prove that, under certain conditions involving an interruption or complete arrest of the current of the fluid through the thoracic duct, the resultant distension and dilatation may prove sufficient to produce rupture, which may take place either in the receptaculum or in the lacteals. In those cases where rupture did not take place, the current of the fluid was re-established through anastomosing branches.

The clinical and post-mortem observations are even more conclusive. They connect directly the process of gradual occlusion of the duct by disease with the concurrent development of a diffuse area of lymphangiectasis, which in some cases terminated in rupture and extravasation.

Monro cites the case reported by Morton of "hydrops ascites lactea" in which the rupture of the lacteals was occasioned by the compression of the thoracic duct near the subclavian vein by large indurated tumors.

Morgagni¹ refers to the cases of Valsalva and Santorino, in both of which there was considerable dilatation of the lacteal vessels produced by the pressure of an aneurismal tumor on the thoracic duct. In Valsalva's case the aorta from the heart to the diaphragm was dilated, and "the lacteal vessels presented the appearance of numerous white spots having a diversity of figure and magnitude."

In the body of a man who died after a large quantity of chylous liquor had been let out of his thorax, Bassius² discovered an orifice at or about the third dorsal vertebra from which the chylous material flowed as from a fountain. Below this orifice the thoracic duct was pervious.

Cayley³ reports a case of obstructed thoracic duct and rupture of the receptaculum chyli. "On post-mortem examination some yellow fluid was found in the peritoneal cavity, and the peritoneum in front of the spine was bulged forward by the effusion behind it of a large quantity of milk-looking fluid. The receptaculum was much dilated, and on its anterior surface was a perforation two inches in length. The thoracic duct was immensely dilated throughout its course to its junction with the subclavian vein, where it suddenly became narrowed, and just at its mouth a fibrinous vegetation was attached to the lining membrane of the vein which completely occluded the opening of the duct. The narrowed part of the duct was blocked by a firm, yellow cylindrical coagulum."

Virchow⁴ refers to the case he observed in a new-born calf, where in consequence of thrombosis of the external jugular vein the mouth of the thoracic duct was occluded, and nearly all the internal organs were dilated to the utmost with ectatic lymph-vessels filled with a slightly sanguinolent fluid. The intestines especially were covered with broad bead-like canals,

¹ 17 Epistle, De Sedibus et Causis Morborum, 1761, Lib. II. p. 152.

² Observationum Decade Secundum, Observatio Septima.

³ Trans. Path. Soc. Lond., vol. xvii. p. 163, 18 6.

⁴ Virchow, Archiv, vol. vii. p. 130.

arranged so closely together that the intervening tissue could scarcely be recognized.

Ormerod¹ reports a case of chylous ascites from which on several different occasions large quantities of a milky fluid were removed. This fluid Drs. Wilks and Marcet proved to be chyle, mixed, more or less, with common serous peritoneal effusion. On post-mortem examination "the left subclavian vein and its afferent vessels were found plugged with a light-colored, ragged clot, evidently of long standing. It was soft in the centre, and at one point was firmly adherent to the walls of the subclavian vein. In the abdomen and chest were also found numerous nodular masses consisting of an imperfectly fibrous structure."

Many other cases might be introduced in which dilatation of the thoracic duct, to a greater or less extent, was the result of partial or complete occlusion near its outlet, caused by the pressure of aneurismal or other tumors, or by disease of the coats of the duct; but such cases have rarely been associated with lymphangiectasia, other than cylindrical dilatation of the duct and enlargement of the receptaculum. In most of such cases the stenotic condition of the duct was compensated by the development of anastomosing branches through which the chyle and lymph channel connection with the venous system was preserved.

The preceding citations of the experimental and clinical data establish, inferentially if not conclusively, the preliminary proposition, that retardation or arrest of the current of the fluid in the thoracic duct at or near its outlet into the subclavian vein may produce dilatation, distension, and rupture of lymphatic vessels, and consequent effusion of chyle and lymph into the serous cavities. The remoteness of the effects from the obstacle interrupting the current of the fluid may depend upon various concomitant conditions, especially upon the anatomical integrity and the tensibility of the vascular walls. When the impediment to the flow of chyle and lymph occurs at or near its outlet the area of stasis and ectasis would necessarily depend upon the duration and extent of such obstacle; and, hence, when the area of ectasia is extensive the manifest inference is that the cause has been one of gradual development and protracted duration. In the case of Bassius, dilatation and rupture were caused by impermeability of the distal extremity of the duct; in Morton's case the rupture of the lacteals was occasioned by the pressure of a tumor upon the duct near the subclavian vein. In Ormerod's case of chylous ascites the interruption to the flow of the chyle was caused by the "plugging of the left subclavian vein and its affluent branches with a light-colored, ragged clot, evidently of long standing." The resistance to the flow of the contents of the thoracic duct must have been derived exclusively from the interrupted venous blood current. The heart was normal and the duct was

¹ Trans. Path. Soc. Lond., vol. xix. p. 199, 1868.

unobstructed. In Cayley's case of obstructed thoracic duct and rupture of the receptaculum, two factors were in operation, interrupted blood-current, occasioned by the fibrinous vegetations attached to the intima of the subclavian vein, and the partial obliteration of the lumen of the duct near its terminus. In Virchow's case of the new-born calf, the condition existing partially exemplifies the obstructing forces operating in the cases of Ormerod and Cayley. The mouth of the duct was occluded by a thrombosis of the external jugular. In addition to the slowing of the venous blood-current in the jugular and in the subclavian, produced by the projecting part of the thrombus, the exit of the fluid from the duct was also impeded. The occlusion of the mouth of the duct was probably inconstant and incomplete during life, nevertheless the area of lymphangiectasis was extensive, involving nearly all the internal organs.

The force exerted upon the contents of the thoracic duct by the passing current of the blood in the subclavian vein is nothing more than an illustration of the hydraulic principle of Venturi; that is, if a stream of water is made to flow through a tube, into which another opens and communicates at its distal end with a reservoir of water, a current will be established in the second tube and continue as long as the stream flows through the first tube, or any fluid remains in the reservoir of the second. The anatomical junction of the thoracic duct with the venous system is peculiarly favorable to the maximum development of this principle, for the near affluence of the internal jugular, and the downward inclination of the duct to the subclavian, contribute to the enhancement of the effect upon the current of chyle. Out of this principle of Venturi must grow the converse, that any retardation of, or obstacle to, the current of blood in the subclavian vein, either from diminution of its lumen or from regurgitant heart affections, must slow the movement of the chyle in the duct and in its tributaries.

The influence of partial obliteration or stenosis of the thoracic duct at or near its termination in the causation of dilatation or rupture of some part of the lymph vascular system cannot be doubted. The evidences of experimentation must be accepted as conclusive. The effects of stenosis produced by the gradual encroachment from disease of the duct or of surrounding and contiguous tissues and structures have been shown by the clinical and *post-mortem* citations. In this latter class of cases the ectasia is gradual in its development and more extensive in its field. In those cases where the lymphangiectasis was consequent upon the slowing or interruption of the venous blood-current in the left subclavian vein, the relation of cause and effect seems equally well established. It remains now to show that such diseases of the heart as slow, impede, or stagnate the venous blood-current in the left subclavian or innominate vein, may also produce lymph-stasis. Petters¹ reports a case of lymphangiectasis in

¹ Vierteljahrschrift für die Practische Heilkunde, vol. xiv. p. 141, 1861.

which were found upon the visceral peritoneum "numerous cysts from the size of peas to that of filberts, some colored bright, and containing serum. Upon the small intestines numerous lenticular protuberances, transparent, and filled with wine-yellow fluid. . . The glands of the right inguinal region were transformed into cysts of small walnut size, filled with wine-yellow fluid. From the inner wall of these cysts extended dilated lymph-vessels of crow-quill size, which connected the cysts with each other, so that a dilated vas efferens and afferens could be seen in each cyst. Upon puncture a yellowish fluid spouted from these cysts in a jet of several inches. The lymph-vessels and cysts formed a mass which it was difficult to unravel. The lymph-vessels in the vicinity and the thoracic duct were greatly dilated.

"The heart occupied the entire anterior thoracic cavity. The enormous enlargement of the heart included the entire right side of the organ, so that the left ventricle with the auricle formed only an appendix. The muscular tissue of the left ventricle, which would not contain a hen's egg, was thin and flaccid. The left venous orifice, by the growing together of the apices of the bicuspid valves, was changed into a small crescentic opening, barely admitting the point of the little finger. The latter was callous and inclosing here and there a bony plate. Right ventricle largely dilated, with thinned walls. Columnæ carneæ of both ventricles very slender and tendinous. Right auricle as large as a child's head, its walls here and there formed of a thin layer of pericardium. Apertures of coronary vein gaping finger wide. Aortic valve competent. Right venous orifice contracted."

This autopsy discloses a stenosis of the left venous orifice, and a moderate contraction of the right ventricular opening. Petters concluded that the stasis of lymph, followed by great dilatation of the lymph-vessels and glands, was caused by the organic disease of the heart. It cannot be doubted that the interruption of the current of blood by the narrowing of the orifices and dilatation of the right side of the heart retarded the movement of the column of fluid in the lymph-vessels. This influence, first demonstrating itself upon the movement of the contents of the thoracic duct, causing distension and dilatation of the trunkal vessels, was transmitted backward, or rather produced stasis of lymph in remote vessels. The lenticular eminences, filled with a transparent fluid, grouped together on the mucous surface of the small intestines, were, probably, ampullar dilatations of terminal lymph-spaces, and the bodies described as cysts on the peritoneum were of like histological nature. Concerning the nature of the gland implication there can be no doubt. The case exemplifies the three varieties of lymphatic telangiectasis made by Lebert, in its lenticular eminences, cyst-like bodies, and transformed glands.

At the conclusion of this report, Petters refers to the case of an old female "suffering with stenosis of the left venous orifices of the heart,"

upon whose right arm he found several semi-solid tumors, situated immediately under a fatless cutis, which he regarded as dilated lymph-glands.

T. Granger Stewart¹ reports a case of "dilatation of the lacteals," associated with an hypertrophied and fatty heart, with diseased aortic valves and dilated auriculo-ventricular orifices. In this case the stasis of chyle occurred in the intestinal villi, producing dilatation of the central lacteal vessels of the villi, and arresting the further absorption of the chyle. To the incompetency of the aortic and auriculo-ventricular valves was added fatty degeneration of the heart. Here enfeebled action of the organ and regurgitation were co-operating factors in slowing the movement of the chyle.

In Rokitsky's² case of chylous ascites, there was dilatation of the heart, "with thickening and shortening of the mitral valves." The cardiac affection not only interrupted the flow of chyle in the thoracic duct, but caused a stasis of chyle in the lacteals and mesenteric glands; of lymph in the subpleural lymphatics; and an effusion of a milk-like fluid into the pleural and peritoneal cavities.

In Hughes's³ case of abdominal effusion of a chylous fluid, supposed to have resulted from the pressure of mesenteric tumors, "the heart was thin, pale, weak, and flabby," and numerous "lacteals—large, tortuous, varicose, and distended, some with milk and others with a clear fluid"—were observed in all parts of the mesentery. The case was complicated with abdominal tumors, "consisting of several agglomerated mesenteric glands," believed to have been cancerous; but from a number of the enlarged glands "a cream-like fluid exuded," and hence it is not improbable that the morbid condition of the glands and lacteals was due to stasis of chyle and lymph, caused by the disease of the heart.

In this connection the case of mitral and tricuspid insufficiency, with granular liver, ascites, anasarca, and thrombosis, reported by Oppolzer,⁴ is interesting. The inferior and superior venæ cavæ were occupied at their outlets with pale, yellow, soft coagula. From the superior, the clot extended with a jagged end into the auricle, and continued into the right subclavian, as well as into the right internal jugular as far as the foramen lacerum. The left innominate and the terminus of the left subclavian were obliterated. The thoracic duct was plugged at its termination by a pale-red, fibrous coagulum. Its walls were thickened, and its lumen, from the receptaculum to the occluding thrombus, was greatly dilated. The regurgitation of blood into the thoracic duct was caused by the obstruction to its entrance into the heart.

There is another class of cases of lymphangiectasis associated with

¹ Edinburgh Med. Journ., vol. ix. p. 448, 1863.

² Path. Anat., Bd. ii. s. 388; also Ziemssen's Cylco., vol. vi. p. 531.

³ Guy's Hospital Reports, vol. vi. p. 297, 1841.

⁴ Allgemeine Wiener Medicinisch. Zeitung, p. 149, 1861.

symptoms of cardiac disease, but the direct connection of the lymph-stasis with an affection of the heart was not proven by a *post-mortem* examination. The following, reported by Cholmeley,¹ is an example of this class :—

This was a case of great enlargement of the right lower extremity, with an intermitting flow of a milky fluid from behind the ankle. The lymphatic engorgement began at the age of three years, and continuously increased during the succeeding six years, when it had involved the whole leg up to the groin. “The pulse was normal as to frequency and rhythm, and of fair volume and force; but all over the heart there was a soft, blowing, systolic murmur, which was loudest at the junction of the second left costal cartilage with the sternum.”

The child was “short, stout, and generally healthy-looking, with a good bright-red color in the cheeks and lips, but was easily affected by cold, and then complained of a want of breath and a feeling of tightness in the chest; and at such times the complexion assumed a markedly livid tint; respiration became somewhat labored and noisy, the extremities cold, and the nails dark-blue.”

The clinical history does not, in the absence of a *post-mortem* examination, positively establish any connection between the lymph-stasis which first appeared near the ankle, and the disturbed cardiac circulation; but such an hypothesis cannot be excluded. The remoteness and limitation of the area of lymphatic engorgement cannot be accepted as negative conditions disproving it. Retardation of the venous current near the heart, and consequent repletion and distension of the thoracic duct and receptaculum, would assuredly offer resistance to the movement of the lymph in affluent vessels. The assumption that the engorgement could have been due to devastated inguinal glands, or occluded lymph-vessels in the abdominal cavity, is discredited by the appearance of the intumescence first about the ankle, and its gradual and continuous extension upward; whereas, if either of those conditions had existed, the lymph-stasis would have begun at the locality of such obstruction and extended in the reverse direction. Lymphatic engorgement does not follow the law of symmetry, and its limitation to one lower extremity may be ascribed to any one or more of the variety of circumstances, such as the varying tensibility, location, distribution, or the anastomosing connections of the lymph vascular system of the lower extremities.

The explanation of this relation of cause and effect between cardiac disturbances and lymph-stasis must, in a general way, be sought in the varying conditions of blood-pressure. A potential, if not the predominant factor concerned in the locomotion of the lymph is the difference between the arterial and venous blood-pressure; the greater this difference the more rapid the movement of the lymph. In a normal condition of the

¹ Trans. Clin. Soc. Lond., vol. 41. p. 116, 1869.

heart, venous orifices, and great afferent and efferent vessels, the blood-pressure in the innominate veins and their affluent branches, during diastole, is at its minimum—represented in comparison with the pressure at the aortic summit, as *nil*; so that, whatever may be the potentiality of the difference between the arterial and venous blood-pressure as a force in promoting the locomotion of the chyle and lymph, it is at its maximum when the venous blood-current is flowing most easily and rapidly into the right auricle. Therefore, it must follow that any disease of the heart, or any condition of its affluent veins which retards, impedes, or obstructs the free and rapid flow of the venous blood into the right auricle and through the tricuspid orifice, will slacken, interrupt, and obstruct the movement of the chyle and lymph in the thoracic duct and its tributary vessels. This conclusion is not derived merely through inductive reasoning, but is illustrated and verified by clinical observation and post-mortem appearances.

The chief function (Küss) of the right auricle is to facilitate the flow of the venous blood by allowing itself to be filled, thereby lessening the blood-pressure in the affluent veins. During its contraction reflux is prevented by the fulness of the veins and by the elasticity of the ventricular walls. If these functions are disturbed and changed by disease of the walls of these cavities, reflux may take place, with increased blood-pressure in the affluent veins, lessening of difference between the arterial and venous blood-pressure, and consequent slowing of the lymphatic current.

If, as claimed by some, the circulation of the lymph is due mainly to transudation and endosmosis taking place at the periphery, increased pressure in the venous system would be equally, if not more effective in the causation of disturbances of the intermediate plasmatic circulation. The inquiry is not, however, into the causes of lymph-stasis and ectasis, but relates to the influence of cardiac disease in the production of these affections of the lymphatic system.

In conclusion I append the following quotations :—

“Obstructive engorgement of the great veins extends also to the thoracic duct. When the subclavian vein is filled to distension, the flow of chyle and lymph must encounter a resistance equal to that opposed to the current of any other vessel which empties into the subclavian. Nay, if lymph be the source of the fibrin in the blood, we see, upon simple physical grounds, why the blood of emphysematous patients is poor in fibrin, why the venous crasis presents hyperinosis and increase of fibrin. Restricted afflux of chyle must, moreover, prejudice nutrition both of the blood and of the entire organism. It is one of several causes which contribute to the general emaciation and to the premature marasmus of emphysematous persons; perhaps, too, it may account for the lack of albumen in the serum of the blood, which produces a tendency to the establishment of drop-sical symptoms.”¹

“The inadequate emptying of the thoracic duct into the left subclavian vein, over-distended with blood, will, in cases of pulmonary emphysema, lead to impoverishment of the blood in so far as concerns such elements as are derived from the lymph, the colorless blood-corpuscles, fibrin, and albumen.”²

¹ Niemeyer, Text-Book of Practical Medicine, vol. i. p. 120.

² Hertz, Ziemssen's Cyclop., vol. v. p. 382.

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